Unraveling the Cause of Avian Vacuolar Myelinopathy in North Carolina

The U.S. Fish and Wildlife Service (Service) and partners recently completed a multi-year investigation of avian vacuolar myelinopathy (AVM), an unusual neurological disease which has killed at least 99 bald eagles since 1994 and thousands of coots wintering in Arkansas, Georgia, North Carolina, and South Carolina. While the cause of AVM remains unknown, Service partnerships with the National Wildlife Health Center, Southeastern Cooperative Wildlife Disease Study, North Carolina State University, North Carolina Wildlife Resources Commission and others have provided advances in our understanding of the disease.

A significant milestone was recently attained with our cooperator's publication* of results from feeding trials aimed at identifying the route of exposure and causative agent of AVM:


AVM-affected American coot

*** Rocke TE, NJ Thomas, CU Meteyer, CF Quist, JR Fischer, T Augspurger and SE Ward. 2005. Attempts to identify the source of avian vacuolar myelinopathy for waterbirds. *Journal of Wildlife Diseases* 41: 163-170.**

Among the direct results of that work are the following observations:

- Two captive-reared mallards that ingested a sample of *Hydrilla verticillata* (along with any material associated with its external surface) from a reservoir where AVM was occurring in wild birds developed brain lesions consistent with AVM.

- Ingestion of numerous other samples of *Hydrilla* from AVM-affected lakes and a lake with no prior history of AVM, other materials from affected lakes (sediments, algae, fish, invertebrates, and water), or tissues from AVM-affected birds did not produce either clinical signs or brain lesions in any of the other test animals in our studies.

- The results suggest that waterbirds are most likely exposed to the causative agent of AVM while feeding on aquatic vegetation, but we do not believe the vegetation itself is the agent. We hypothesize that the agent might either be accumulated by aquatic vegetation, such as *Hydrilla,* or associated with biotic or abiotic material on its external surfaces.

The Southeastern Cooperative Wildlife Disease Study and Clemson University have reproduced the brain lesions characteristic of AVM through feeding material from lakes with AVM-positive birds to coots, ducks, hawks, and chickens. While feeding birds hydrilla from affected lakes has resulted in transfer of the disease, others have demonstrated that: 1) hydrilla does need not be present for AVM to occur, 2) AVM does not occur at many lakes with hydrilla (which is extremely common in the southeast despite the disease being known from less than 15 impoundments), 3) experimental birds do not develop AVM lesions after consuming hydrilla from AVM-negative (i.e., control) lakes, and 4) experimental birds do not always develop AVM lesions after eating hydrilla from AVM-positive lakes.

Other results of our study provide a better scientific understanding of the species affected, the timing and duration of the effects, and the magnitude of impacts:

- Typically, clinical disease is noticed in the autumn or early winter—as early as October at some lakes—where sick birds are observed for several months afterwards.
- Exposure to the causative agent of AVM is site-specific and seasonal.
- Onset of disease can be rapid, within as little as 5 days post-exposure.
- AVM, once thought to affect only coots and eagles, has been documented in a variety of birds, and mallards can be used effectively as sentinels to monitor the disease.

We've used sound science and cooperation to study this disease with very limited direct funding. Our expertise and equipment in the field, close to where the birds are affected, makes us a valuable partner for scientists with a more national or regional focus. This is just one example of the importance of our field presence in facilitating wildlife assessment and management.

The current paper and others associated with the study (see list below) can be obtained from the USFWS at http://nc-es.fws.gov/ecotox/. A list of current AVM-related publications by others can also be obtained at the website. For more information, contact Tom Augspurger, U.S. Fish and Wildlife Service, PO Box 33726, Raleigh, NC 27636-3726 (919/856-4520 ext 21 or tom_augspurger@fws.gov).

Augspurger T, JR Fischer, NJ Thomas, L Sileo, RE Brannian, KJG Miller and TE Rocke. 2003. Vacuolar myelinopathy in waterfowl from a North Carolina impoundment. *Journal of Wildlife Diseases* 39: 412-417.

Dodder NG, B Strandberg, T Augspurger and RA Hites. 2003. Lipophilic organic compounds in lake sediment and American coot (*Fulica americana*) tissues, both affected and unaffected by avian vacuolar myelinopathy. *Science of the Total Environment* 311: 81-89.

Larsen RS, FB Nutter, T Augspurger, TE Rocke, NJ Thomas and MK Stoskopf. 2003. Failure to transmit avian vacuolar myelinopathy to mallard ducks. *Journal of Wildlife Diseases* 39: 707-711.

Larsen RS, FB Nutter, T Augspurger, TE Rocke, L Tomlinson, NJ Thomas and MK Stoskopf. 2002. Clinical features of avian vacuolar myelinopathy in American coots. *Journal of the American Veterinary Medical Association* 221: 80-85.

Rocke TE, NJ Thomas, T Augspurger and K Miller. 2002. Epizootiologic studies of avian vacuolar myelinopathy in waterbirds. *Journal of Wildlife Diseases* 38: 678-684.